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SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R) 1950-2007/Aug 17
         (c) format only 2007 Dialog
  File  55:Biosis Previews(R) 1993-2007/Aug W2
         (c) 2007 The Thomson Corporation
  File  34:SciSearch(R) Cited Ref Sci 1990-2007/Aug W3
         (c) 2007 The Thomson Corp
  File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
         (c) 2006 The Thomson Corp

      Set  Items  Description
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? s pRB or (retinoblastoma(w)susceptibl?(w)gene)
           10365  PRB
           46661  RETINOBLASTOMA
          218136  SUSCEPTIBL?
         3140356  GENE
              12  RETINOBLASTOMA(W)SUSCEPTIBL?(W)GENE
      S1   10377  PRB OR (RETINOBLASTOMA(W)SUSCEPTIBL?(W)GENE)
? s transcriptional(w)factor??
          327353  TRANSCRIPTIONAL
         5555631  FACTOR??
      S2    7083  TRANSCRIPTIONAL(W)FACTOR??
? s s1 and s2
           10377  S1
            7083  S2
      S3      19  S1 AND S2
? rd
      S4      10  RD  (unique items)
? t s4/3,k,ab/1-10

 4/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2007 Dialog. All rts. reserv.
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16271647 PMID: 17015480

TReP-132 is a novel progesterone receptor coactivator required for the inhibition of breast cancer cell growth and enhancement of differentiation by progesterone.

Gizard Florence; Robillard Romain; Gross Barbara; Barbier Olivier; Revillion Francoise; Peyrat Jean-Philippe; Torpier Gerard; Hum Dean W; Staels Bart

INSERM U545, Institut Pasteur de Lille, 1 rue Calmette, BP 245, 59019 Lille, France.

Molecular and cellular biology (United States) Oct 2006, 26 (20) p7632-44, ISSN 0270-7306--Print Journal Code: 8109087

Publishing Model Print

Document type: Journal Article; Research Support, Non-U.S. Gov't

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The sex steroid progesterone is essential for the proliferation and differentiation of the mammary gland epithelium during pregnancy. In relation to this, in vitro studies using breast carcinoma T47D cells have demonstrated a biphasic progesterone response, consisting of an initial proliferative burst followed by a sustained growth arrest. However, the transcriptional factors acting with the progesterone receptor (PR) to mediate the progesterone effects on mammary cell growth and differentiation remain to be determined. Recently, it has been demonstrated that the transcriptional regulating protein of 132 kDa (TReP-132), initially identified as a regulator of steroidogenesis, is also a cell growth suppressor. Similar to progesterone-bound PR, TReP-132 acts by inducing the gene expression of the G1 cyclin-dependent kinase inhibitors p21WAF1/Cip1 (p21) and p27Kip1 (p27). The putative interaction between

TReP-132 and progesterone pathways in mammary cells was therefore analyzed in the present study. Our results show that TReP-132 interacts in vitro and in T47D cells with progesterone-activated PR. TReP-132 synergizes with progesterone-bound PR to trans activate the p21 and p27 gene promoters at proximal Sp1-binding sites. Moreover, TReP-132 overexpression and knockdown, respectively, increased or prevented the induction of p21 and p27 gene expression by progesterone. As a consequence, TReP-132 knockdown also resulted in the loss of the inhibitory effects of progesterone on pRB phosphorylation, G1/S cell cycle progression, and cell proliferation. Furthermore, the knockdown of TReP-132 expression also prevented the induction of both early and terminal markers of breast cell differentiation which had been previously identified as progesterone target genes. As well, the progesterone-induced accumulation of lipid vacuoles was inhibited in the TReP-132-depleted cells. Finally, TReP-132 gene expression levels increased following progesterone treatment, indicating the existence of a positive auto-regulatory loop between PR and TReP-132. Taken together, these data identify TReP-132 as a coactivator of PR mediating the growth-inhibitory and differentiation effects of progesterone on breast cancer cells.

... response, consisting of an initial proliferative burst followed by a sustained growth arrest. However, the ***transcriptional*** ***factors*** acting with the progesterone receptor (PR) to mediate the progesterone effects on mammary cell growth...

... TReP-132 knockdown also resulted in the loss of the inhibitory effects of progesterone on pRB phosphorylation, G1/S cell cycle progression, and cell proliferation. Furthermore, the knockdown of TReP-132...

4/3,K,AB/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

16030392 PMID: 16510138
Involvement of the transcriptional factor E2F1 in the regulation of the rRNA promoter.
Ayrault Olivier; Andrique Laetitia; Seite Paule
Laboratoire d'Oncologie Moleculaire, EA 3805, Pole Biologie-Sante, 40, avenue du Recteur Pineau, 86022 Poitiers cedex, France.
Experimental cell research (United States) Apr 15 2006, 312 (7) p1185-93, ISSN 0014-4827--Print Journal Code: 0373226
Publishing Model Print-Electronic
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
p16INK4a-pRB -E2F and ARF-MDM2-p53 are two major tumor suppressor networks involved in cell proliferation control. The nucleolar ARF protein binds to MDM2 to activate the growth suppressive functions of p53, but can also exert its tumor suppressor activity independently of p53, through mechanisms involving other regulators: in that manner, p14ARF has been shown to inhibit the transcriptional activity of E2F1 in vitro, suggesting that the two pathways intersect with one another. More recently, ARF has been shown to inhibit ribosomal RNA processing, and to specifically interact with the rRNA promoter, suggesting a role in the regulation of both maturation and transcription processes. We show here that E2F1 can bind the rRNA promoter and modulate its activity through the interaction with two E2F1-binding sequences we have identified. The regulation of ribosome biogenesis appears as a major p53-independent process, which involves both ARF and E2F1 to control cell proliferation.

Involvement of the transcriptional factor E2F1 in the regulation of the rRNA promoter.

p16INK4a-pRB -E2F and ARF-MDM2-p53 are two major tumor suppressor networks involved in cell proliferation...

4/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

12378728 PMID: 10223185
HGF-mediated apoptosis via p53/bax-independent pathway activating JNK1.
Conner E A; Teramoto T; Wirth P J; Kiss A; Garfield S; Thorgeirsson S S
Laboratory of Experimental Carcinogenesis, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892, USA.
Carcinogenesis (ENGLAND) Apr 1999, 20 (4) p583-90, ISSN 0143-3334--Print Journal Code: 8008055
Publishing Model Print
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed
Current studies have indicated both positive and negative roles for the hepatocyte growth factor (HGF)/c-met receptor signaling system in tumor development. Recently, we have shown that HGF has the capacity to induce both growth inhibition and programmed cell death in aflatoxin-transformed (AFLB8) rat liver epithelial cells. Using the same cell line, we have now investigated a potential mechanism for HGF-induced apoptosis. Immunoblot analysis of bcl-2 gene family member (bax, bcl-2, bclX-s/l) expression showed no correlation with HGF treatment, suggesting that HGF-mediated apoptosis is bax independent. Following HGF treatment retinoblastoma protein (***pRB***) was present in the hypophosphorylated state. HGF treatment increased cyclin A, cyclin G1 and nuclear transcriptional ***factor*** (NFkappaB) protein expression. However, electrophoretic mobility shift analysis showed that NFkappaB activity decreased with HGF treatment. Under these apoptotic conditions, c-Jun N-terminal kinase (JNK1) and extracellular signal-regulated kinase (ERK2) were activated with lower level activation of ERK2, while no involvement of phosphatidylinositol-3 kinase was observed. Epidermal growth factor (EGF) was not protective, and actually induced cells to undergo apoptosis to a level similar to that of HGF alone or EGF/HGF in combination. These results suggest the possibility of cross-talk between HGF/c-met and EGF/EGFR signaling pathways, and the involvement of JNK1 induction in HGF-mediated apoptotic cell death.

... HGF treatment, suggesting that HGF-mediated apoptosis is bax independent. Following HGF treatment retinoblastoma protein (***pRB***) was present in the hypophosphorylated state. HGF treatment increased cyclin A, cyclin G1 and nuclear transcriptional factor (NFkappaB) protein expression. However, electrophoretic mobility shift analysis showed that NFkappaB activity decreased with HGF...

4/3,K,AB/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

11715516 PMID: 9515032
Changes in E2F complexes containing retinoblastoma protein family members and increased cyclin-dependent kinase inhibitor activities during terminal differentiation of cardiomyocytes.
Flink I L; Oana S; Maitra N; Bahl J J; Morkin E
University Heart Center and Department of Medicine, University of Arizona, Tucson, AZ 85724, USA.
Journal of molecular and cellular cardiology (ENGLAND) Mar 1998, 30 (3) p563-78, ISSN 0022-2828--Print Journal Code: 0262322
Contract/Grant No.: POHL 20984; HL; NHLBI

Publishing Model Print
Document type: In Vitro; Journal Article; Research Support, Non-U.S. Gov't; Research Support, U.S. Gov't, P.H.S.
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

Cardiomyocyte terminal differentiation was examined by studying the interaction of retinoblastoma protein (pRb) family members with E2F during the developmental transition from 17-day fetal to 2-day neonatal. Additionally, the expression pattern of cyclins, cyclin-dependent kinases (CDKs), and CDK inhibitors responsible for modulating the phosphorylation of ***pRb*** were studied. p107, ***pRb*** , and p130 are regulators of cellular proliferation, differentiation, and cell cycle exit and entry, respectively. The active, underphosphorylated form of these proteins targets the E2F family of transcriptional factors that play a critical role in the control of genes associated with DNA synthesis. Electromobility shift analyses demonstrated E2F complexed with p107 in proliferating fetal cardiomyocytes, whereas in 2-day neonatal cells, E2F was principally associated with p130 and a low level of ***pRb*** . At the 2-day neonatal stage, decreased protein levels were observed for cyclins D2, D3, and E, and CDK2 and CDK4. No changes were observed in the mRNA levels of the D-cyclins in neonatal cells; however, the transcripts for cyclins A and E and CDK4 were diminished. In skeletal myoblasts, differentiation is associated with induction of p21, a CDK inhibitor, by a MyoD-dependent pathway. Although heart cells lack MyoD, CDK assays demonstrated that the activity of CDKs 2, 4, and 6 were downregulated in 2-day neonatal cells, and CDC2 was increased. RT-PCR indicated that p21 mRNA was induced 1.4-, 2.0-, and 3.1-fold in the 2-day neonatal, 7-day neonatal, and adult stages, respectively, compared to the 17-day fetal stage. At the protein level, p21 also increased at the 2-day neonatal stage. Kinase inhibitory immunodepletion assays showed that CDK inhibitory activity was markedly increased in the 2-day neonate. Although mRNA levels of the p27 CDK inhibitor were unchanged, its protein level and inhibitory effect on CDK2 and CDK4 were increased. Thus, cardiomyocytes retain the capacity to proliferate until the early neonatal period when a series of changes occur, including a switch in pRb partners, a decrease in CDK levels and induction of CDK inhibitory activity, which is associated with terminal differentiation.

Cardiomyocyte terminal differentiation was examined by studying the interaction of retinoblastoma protein (pRb) family members with E2F during the developmental transition from 17-day fetal to 2-day...

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...2-day neonatal cells, E2F was principally associated with p130 and a low level of ***pRb*** . At the 2-day neonatal stage, decreased protein levels were observed for cyclins D2, D3...
... until the early neonatal period when a series of changes occur, including a switch in pRb partners, a decrease in CDK levels and induction of CDK inhibitory activity, which is associated...

4/3,K,AB/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10738325 PMID: 8845642

The retinoblastoma gene product negatively regulates cellular or viral oncogene promoters in vivo.

Salcedo M; Garrido E; Taja L; Gariglio P
Unidad de Investigacion Medica en Enfermedades Oncologicas, Hospital de Oncologia, Centro Medico Nacional Siglo XXI, Instituto Mexicano del Seguro Social, Mexico, D.F.

Archives of medical research (MEXICO) 1995, 26 Spec No pS157-62, ISSN 0188-4409--Print Journal Code: 9312706
Publishing Model Print
Document type: Journal Article; Research Support, Non-U.S. Gov't
Languages: ENGLISH
Main Citation Owner: NLM
Record type: MEDLINE; Completed

The protein product of the retinoblastoma tumor suppressor gene (pRb) has been demonstrated to bind transcriptional ***factors*** such as E2F and c-Myc protein in vitro. To determine whether pRb regulates both cellular (c-myc) and viral (HPV LCR) promoter activity in vivo, MYC-CAT or HPV LCR-CAT chimeric expression plasmids were generated and cotransfected with a pCMV-RB expression plasmid. ***pRb*** repressed both myc and LCR transcription but not SV40-CAT. Transcriptional repression induced by pCMV-RB was relieved by addition of pSV2-E7. Moreover, immunohistochemical assays indicate that in cervical intraepithelial neoplasia lesions, an increased pRb expression correlates with decreased c-myc oncogene expression. These results suggest that pRb can negatively regulate c-myc transcription in vivo in both normal tissue or early cervical lesions. However, in HPV induced invasive cervical carcinomas where viral DNA is integrated expressing its oncoproteins, pRb could be complexed by HPV E7 oncoprotein releasing the repression effect and promoting cell growth.

The protein product of the retinoblastoma tumor suppressor gene (pRb) has been demonstrated to bind transcriptional ***factors*** such as E2F and c-Myc protein in vitro. To determine whether pRb regulates both cellular (c-myc) and viral (HPV LCR) promoter activity in vivo, MYC-CAT...

... LCR-CAT chimeric expression plasmids were generated and cotransfected with a pCMV-RB expression plasmid. ***pRb*** repressed both myc and LCR transcription but not SV40-CAT. Transcriptional repression induced by pCMV ...

... of pSV2-E7. Moreover, immunohistochemical assays indicate that in cervical intraepithelial neoplasia lesions, an increased pRb expression correlates with decreased c-myc oncogene expression. These results suggest that pRb can negatively regulate c-myc transcription in vivo in both normal tissue or early cervical...

... However, in HPV induced invasive cervical carcinomas where viral DNA is integrated expressing its oncoproteins, pRb could be complexed by HPV E7 oncoprotein releasing the repression effect and promoting cell growth.

4/3,K,AB/6 (Item 1 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

14211478 BIOSIS NO.: 199800005725
Intergenic interrelationships in cell cycle regulation: Key role of the E2F family transcriptional factors

AUTHOR: Kel' O V (Reprint); Kel A E
AUTHOR ADDRESS: Inst. Cytol. Genet., Sib. Div., Russ. Acad. Sci., Novosibirsk 630090, Russia**Russia

JOURNAL: Molekulyarnaya biologia (Moscow) 31 (4): p656-670 1997 1997
MEDIUM: print

ISSN: 0026-8984
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Russian

ABSTRACT: E2F factors provide coordinated expression of protein-encoding genes necessary for the cell cycle such as cyclin and cyclin-dependent kinase genes, transcriptional factor E2F- 1 and tumor suppressor RBI genes, genes of some DNA replication and repair enzymes, and some histone genes. The level of the transcription of these genes reaches its maximum during the G1/S transition and at the beginning of the S-phase. Tumor suppressors p53 and ***pRB*** belong to negative regulators of the cell cycle. These proteins regulate the function of E2F factors by direct protein-protein interactions and through a complex gene network of cell cycle regulation. Regulatory regions of 35 genes participating in cell cycle regulation are described in detail in the CYCLE-TRRD database. The mechanisms of E2F binding site function are considered as are the structures of the regulatory regions of E2F-dependent genes and intergenic interrelationships in cell cycle regulation.

Intergenic interrelationships in cell cycle regulation: Key role of the E2F family transcriptional factors

...ABSTRACT: encoding genes necessary for the cell cycle such as cyclin and cyclin-dependent kinase genes, transcriptional factor E2F- 1 and tumor suppressor RBI genes, genes of some DNA replication and repair enzymes...

...G1/S transition and at the beginning of the S-phase. Tumor suppressors p53 and ***pRB*** belong to negative regulators of the cell cycle. These proteins regulate the function of E2F...
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: ***pRB*** gene...

... ***transcriptional*** ***factor***

4/3,K,AB/7 (Item 2 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

14067574 BIOSIS NO.: 199799701634
The interaction between 73 kDa heat shock protein (HSC73) and retinoblastoma protein (pRb): The identification of the binding region of pRb and the function of HSC73
AUTHOR: Sogahata Katsuya (Reprint); Torigoe Toshihiko; Sato Noriyuki
AUTHOR ADDRESS: Dep. Traumatol. Critical Care Med., Sapporo Med. Univ., Sch. Med., Sapporo, Japan**Japan
JOURNAL: Sapporo Medical Journal 66 (1-2): p19-27 1997 1997
ISSN: 0036-472X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: Japanese

ABSTRACT: Retinoblastoma protein (pRb) interacts with transcriptional factors and, particulary, dephosphorylated ***pRb*** functions as a negative regulator of cell cycle. We have previously demonstrated that dephosphorylated pRb was associated with 73kDa heat shock cognate protein (HSC73) in certain tumor cell lines. In this experiment, we analyzed the interaction between these two proteins in vitro and determined the HSC73-binding region of pRb by using GST-deletion mutant pRb fusion proteins and synthetic

peptides corresponding to the amino acid sequence of ***pRb*** . Our data showed that HSC73 interacted directly with a novel region which was located in N-terminal 328-340 amino acid residues outside the pocket region of ***pRb*** . Furthermore, we analyzed the function of HSC73 in its interaction with ***pRb*** in vitro. The analysis using native polyacrylamide gel electrophoresis indicated that the HSC73 could confer the conformational change on pRb, and might protect the aggregation. Dephosphorylated ***pRb*** , but not phosphorylated ***pRb*** was degraded by the liver-derived cytosolic extract when the interaction with HSC73 was blocked by the synthetic peptide. These data suggest that HSC73 acts as the molecular chaperone selectively for the dephosphorylated pRb, thereby potentiating the pRb function as the inhibitory regulator of cell cycle and, further, cell proliferation.

The interaction between 73 kDa heat shock protein (HSC73) and retinoblastoma protein (pRb): The identification of the binding region of pRb and the function of HSC73

ABSTRACT: Retinoblastoma protein (pRb) interacts with transcriptional factors and, particulary, dephosphorylated ***pRb*** functions as a negative regulator of cell cycle. We have previously demonstrated that dephosphorylated pRb was associated with 73kDa heat shock cognate protein (HSC73) in certain tumor cell lines. In...

...the interaction between these two proteins in vitro and determined the HSC73-binding region of pRb by using GST-deletion mutant pRb fusion proteins and synthetic peptides corresponding to the amino acid sequence of ***pRb*** . Our data showed that HSC73 interacted directly with a novel region which was located in N-terminal 328-340 amino acid residues outside the pocket region of ***pRb*** . Furthermore, we analyzed the function of HSC73 in its interaction with ***pRb*** in vitro. The analysis using native polyacrylamide gel electrophoresis indicated that the HSC73 could confer the conformational change on pRb, and might protect the aggregation. Dephosphorylated ***pRb*** , but not phosphorylated pRb was degraded by the liver-derived cytosolic extract when the interaction with HSC73 was blocked...

...peptide. These data suggest that HSC73 acts as the molecular chaperone selectively for the dephosphorylated pRb, thereby potentiating the pRb function as the inhibitory regulator of cell cycle and, further, cell proliferation.

4/3,K,AB/8 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

16327989 Genuine Article#: 152DG Number of References: 148
Title: Conserved functions of retinoblastoma proteins: From purple retina to green plant cells (ABSTRACT AVAILABLE)
Author(s): Miskolczi P; Lendvai A; Horvath GV; Pettko-Szandtner A; Dudits D (REPRINT)
Corporate Source: Hungarian Acad Sci,Inst Plant Biol, Biol Res Ctr,POB 521/H-6701 Szeged//Hungary/ (REPRINT); Hungarian Acad Sci,Inst Plant Biol, Biol Res Ctr,H-6701 Szeged//Hungary/
Journal: PLANT SCIENCE, 2007, V172, N4 (APR), P671-683
ISSN: 0168-9452 Publication date: 20070400
Publisher: ELSEVIER IRELAND LTD, ELSEVIER HOUSE, BROOKVALE PLAZA, EAST PARK SHANNON, CO, CLARE, 00000, IRELAND
Language: English Document Type: REVIEW
Abstract: The mammalian retinoblastoma susceptibility gene product, known

as the first tumor suppressor protein (pRB), has a central role in the regulation of the cell cycle, differentiation and apoptotic pathways of specific cell types. Discoveries in the past decade have shown that key elements of the RB regulatory network also exist in higher plants which control a wide range of cellular functions, including cell division cycle and differentiation. As we outline in this review, the plant RB-related proteins (RBRs) display amino acid sequence similarity and biochemical binding properties analogous to their mammalian homologues and they can interact with E2F ***transcriptional*** ***factors*** , D-type cyclins and viral proteins. The complex regulatory functions of the retinoblastoma proteins are discussed in detail by focusing in particular on the increasing amount of information being produced about the role of these proteins in higher plants.

Abstract: The mammalian retinoblastoma susceptibility gene product, known as the first tumor suppressor protein (pRB), has a central role in the regulation of the cell cycle, differentiation and apoptotic pathways...

...and biochemical binding properties analogous to their mammalian homologues and they can interact with E2F transcriptional ***factors*** , D-type cyclins and viral proteins. The complex regulatory functions of the retinoblastoma proteins are...

4/3,K,AB/9 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

11262498 Genuine Article#: 630KW Number of References: 28
Title: Increase expression of gene of differentiation - E2F4 caused by sulindac derivatives in vitro correlated with apoptosis (ABSTRACT AVAILABLE)
Author(s): Rudzki S (REPRINT) ; Korszen-Pilecka I; Dudzisz-Sledz M; Wojcierowski J
Corporate Source: Med Univ Lublin,Dept Gen & Transplant Surg 1,PL-20950 Lublin//Poland/ (REPRINT); Med Univ Lublin,Dept Gen & Transplant Surg 1,PL-20950 Lublin//Poland/; Med Univ Lublin,Dept Human Genet,PL-20950 Lublin//Poland/
Journal: BULLETIN OF THE VETERINARY INSTITUTE IN PULAWY, 2002, S, P183-189
ISSN: 0042-4870 Publication date: 20020000
Publisher: NATL VETERINARY RESEARCH INST, C/O PUBICATIONS OFFICE, 24-100 PULAWY, POLAND
Language: English Document Type: ARTICLE
Abstract: E(2)F4 gene is a differentiation gene of the cell and the very early transcriptional factor for other genes including E(2)F4 and genes of apoptosis. Overexpression of E(2)F4 gene or decrease of pRB cause cell fast entering S phase of cell cycle and apoptosis. In the presented paper we determine the influence of sulindac sulfide and sulfone derivatives on E(2)F4 expression and apoptosis in LS180 colon carcinoma cells.

Abstract: E(2)F4 gene is a differentiation gene of the cell and the very early transcriptional factor for other genes including E(2)F4 and genes of apoptosis. Overexpression of E(2)F4 gene or decrease of pRB cause cell fast entering S phase of cell cycle and apoptosis. In the presented paper...

4/3,K,AB/10 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

11215818 Genuine Article#: 624VR Number of References: 31
Title: Immunohistochemical expression of the transcription factor DP-1 and its heterodimeric partner E2F-1 in non-Hodgkin lymphoma (ABSTRACT AVAILABLE)
Author(s): Chan JA; Olvera M; Lai R; Naing W; Rezk SA; Brynes RK (REPRINT)

Corporate Source: Univ So Calif,Los Angeles Cty Med Ctr, Dept Pathol,2011 Zonal Ave,HMR 209/Los Angeles//CA/90033 (REPRINT); Univ So Calif,Los Angeles Cty Med Ctr, Dept Pathol,Los Angeles//CA/90033; Univ Texas,MD Anderson Canc Ctr,Houston//TX/77030
Journal: APPLIED IMMUNOHISTOCHEMISTRY & MOLECULAR MORPHOLOGY, 2002, V10, N4 (DEC), P322-326
ISSN: 1062-3345 Publication date: 20021200
Publisher: LIPPINCOTT WILLIAMS & WILKINS, 530 WALNUT ST, PHILADELPHIA, PA 19106-3621 USA
Language: English Document Type: ARTICLE
Abstract: DP-1 is a G1 cell cycle-related protein that forms heterodimers with E2F, a family of transcriptional factors regulating the expression of genes important for G1 to S progression. Although the exact role of DP-1 is not well understood, it has been shown to stabilize DNA binding of E2F proteins. By immunohistochemistry, the authors examined the expression of DP-1 in lymphoid tissues, including 8 cases of reactive follicular hyperplasia and 69 cases of B-cell non-Hodgkin lymphoma. The expression of the cell cycle-related proteins E2F-1 and Ki-67 was also assessed. Scoring was based on the proportion of labeled nuclei (1-10%, 11-25%, 26-50%, and > 50%). In reactive follicular hyperplasia, staining for DP-1, E2F-1, and Ki-67 was largely confined to the germinal centers. All 25 cases of follicular lymphoma, regardless of grade, had a high proportion (> 50%) of DP-1-positive cells but a lower proportion of cells marking for E2F-1 and Ki-67 ($P < 0.001$). The diffuse large B-cell lymphomas ($n = 24$) had high DP-1 and Ki-67 scores but low E2F-1 scores ($P < 0.001$). Small lymphocytic ($n = 10$), marginal zone ($n = 3$), and mantle cell lymphomas ($n = 5$) contained relatively low proportions of cells labeled for all three markers. Precursor B-cell lymphoblastic lymphoma ($n = 2$) displayed high proportions of cells positive for DP-1, Ki-67, and E2F-1 (> 50% in both cases). Except in follicular center cell lesions, DP-1 expression generally correlated with that of Ki-67. However, the expression of DP-1 was discordant with that of E2F-1 in benign and malignant follicular center cells, suggesting that DP-1 may have functions other than facilitating E2F-1-dependent gene regulation and cell cycle progression in these neoplasms.
...Abstract: is a G1 cell cycle-related protein that forms heterodimers with E2F, a family of transcriptional factors regulating the expression of genes important for G1 to S progression. Although the exact role...
...Identifiers--CELL-CYCLE PROGRESSION; MOLECULAR-CLONING; GENE-EXPRESSION; BINDING PROTEIN; S-PHASE; FAMILY; MEMBER; PRB; FIBROBLASTS; COMPONENT
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? s prb or (retinoblastoma (w)susceptibil? (w)gene)
           10365  PRB
           46661  RETINOBLASTOMA
          406648  SUSCEPTIBIL?
         3140356  GENE
            1375  RETINOBLASTOMA(W)SUSCEPTIBIL?(W)GENE
      S1   11400  PRB OR (RETINOBLASTOMA (W)SUSCEPTIBIL? (W)GENE)
? s e2f?
      S2   13588  E2F?
? s s1 and s2
           11400  S1
           13588  S2
      S3    2743  S1 AND S2
? s review
      S4 1313708  REVIEW
? s s3 and s4
            2743  S3
         1313708  S4
      S5     143  S3 AND S4
? rd
      S6      99  RD  (unique items)
? t s6/3,k,ab/1-10

 6/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2007 Dialog. All rts. reserv.

23971228   PMID: 16914062
   E2F1  activation  is responsible for pituitary adenomas induced by
HMGA2 gene overexpression.
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504282 PMID: 15892617

Modulation of pRB/E2F functions in the regulation of cell cycle and in cancer.

Seville Lucy L; Shah Nita; Westwell Andrew D; Chan Weng C

School of Pharmacy, The Centre for Biomolecular Sciences, University of Nottingham, University Park, Nottingham NG7 2RD, UK.

Current cancer drug targets (Netherlands) May 2005, 5 (3) p159-70, ISSN 1568-0096--Print Journal Code: 101094211

Publishing Model Print

Document type: Journal Article; Review

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Cell proliferation is regulated by the cell cycle, and in order to divide the cell must enter a mitotic state. Prior to mitosis the cell is required to pass through a number of checkpoints, including the critical G1/S restriction point governed by the successive phosphorylation of the retinoblastoma protein, ***pRb*** . The various proteins and regulatory factors governing pRb phosphorylation have been a major focus of study in recent years, given the central importance of G1/S transition deregulation in cancer development. This ***review*** summarises the molecular biology around the G1/S transition, focussing on the critical roles of the transcription factor family E2F and the cyclin-dependent kinase (CDK) and cyclin families involved in E2F release from ***pRb*** . Interestingly, ***E2F*** release from ***pRb*** is associated with cell proliferation; however, above a certain threshold E2F has the potential to trigger apoptosis. The ***review*** focuses on the following topics: (i) how E2F and other substrates bind to pRb at the molecular level; (ii) mechanisms by which pRb function is modulated within the cell; (iii) mechanisms that inhibit or enhance cell proliferation via the pRb/E2F pathway; (iv) how E2F can potentiate apoptotic pathways; and (v) what controls whether E2F mediates cell proliferation or apoptosis. The case for the development of agents that perturb pRb:E2F interactions will be made, as a strategy to further inform the molecular biology around this important target and as a therapeutic strategy against cancer.

Modulation of pRB/E2F functions in the regulation of cell

Rb family proteins as modulators of gene expression and new aspects regarding the interaction with chromatin remodeling enzymes.

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The pRb family proteins (pRb1/105, p107, pRb2/p130), collectively referred to as pocket proteins, are believed to function primarily as regulators of the mammalian cell cycle progression, and suppressors of cellular growth and proliferation. In addition, different studies suggest that these pocket proteins are also involved in development and differentiation of various tissues. Several lines of evidence indicate that generally pRb -family proteins function through their effect on the transcription of ***E2F*** -regulated genes. In fact, each of Rb family proteins binds to distinct members of the E2F transcription factors, which regulate the expression of genes whose protein products are necessary for cell proliferation and to drive cell-cycle progression. Nevertheless, pocket proteins can affect the G1/S transition through E2F -independent mechanisms. More recently, a broad range of evidences indicate that pRb -family proteins associate with a wide variety of transcription factors and chromatin remodeling enzymes forming transcriptional repressor complexes that control gene expression. This review focuses on the complex regulatory mechanisms by which ***pRb*** -family proteins tell genes when to switch on and off.